CASE REPORT
Urooncology

Doi: 10.4274/jus.galenos.2025.2025-5-3
J Urol Surg

A Rare Case of Ureteral Metastasis from an Occult Pancreatic Primary

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Abstract

Metastatic involvement of the ureter is one of the rare causes of ureteral obstruction, with most documented cases being identified postmortem. The ureter's anatomy contributes to its natural resistance against metastatic deposits. While malignancies originating in pelvic organs, like the prostate, cervix, and bladder, are the most frequent primary sources of these tumors, metastasis from a pancreatic primary is extremely rare, with only a few cases documented in the literature to date. A 54-year-old male presented with a ureteric stricture. Although initial biopsy revealed a chronic inflammatory stricture, subsequent investigation pointed towards adenocarcinoma originating from the pancreaticobiliary tract. Metastasis-related ureteral obstruction usually occurs a year after primary cancer diagnosis. It is rare for this obstruction to be the only sign of underlying malignancy. In case of an occult primary, positron emission tomography scans and immunohistochemistry are essential for accurate diagnosis. Whether the primary tumor is known or occult, ureteral metastasis indicates advanced disease and a poor prognosis.

Keywords: Adenocarcinoma, metastasis, pancreas, stricture, ureter

Introduction

Ureteral obstruction due to metastatic disease is a rare clinical occurrence (1,2). While approximately 400 cases have been documented in the literature, the majority are identified postmortem (1,3). Ureteral metastasis originating from a pancreaticobiliary primary is particularly uncommon ureteral metastasis originating from a pancreaticobiliary primary; only a few such cases have been previously reported (4).

Case Presentation

A 54-year-old male presented with a two-month history of left lower back pain and significant weight loss (10 kg over two months). He had no history of urinary tract infections, pelvic radiotherapy, nephrolithiasis, or prior ureteral intervention. He had a prior hospitalization at an outside institution where impaired renal function (creatinine 1.95 mg/dL) was noted. Urine cultures were negative. Non-contrast computed tomography (CT) kidneys, ureters and bladder demonstrated left-sided hydronephrosis and hydroureter extending to the iliac vessels. He underwent left-sided double-J (DJ) stenting. While his serum creatinine level improved to 1.2 mg/dL, his back pain persisted.

Following admission at our institute, he underwent further evaluation. Magnetic resonance imaging of the abdomen revealed a left-sided DJ stent *in situ* with minimal renal pelvic fullness, without any features of retroperitoneal fibrosis. The pancreas appeared moderately atrophic. The possibility of genitourinary tuberculosis, a common cause of ureteric stricture in the Indian subcontinent, was also investigated. The urine acid-fast bacilli smear and QuantiFERON-TB Gold tests were both negative.

A left retrograde pyelogram demonstrated a narrow, streak-like flow of contrast medium extending from the L4 to L5 vertebral level, with proximal ureteral dilatation. Ureteroscopy revealed thickened ureteral mucosa from which a biopsy was taken. Histopathological analysis of the specimen revealed chronic inflammation.

Due to persistent symptoms and a left ureteric stricture confirmed on ureteroscopy, the patient underwent a retroperitoneal exploration. Intraoperatively, dense adhesions were found obstructing the left ureter between the L4 and L5 vertebral

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Received: 06.05.2025 **Accepted:** 18.08.2025 **Epub:** 20.08.2025

Cite this article as: Jana S, Raghuvanshi K, Patel V, Chaudhari R. A rare case of ureteral metastasis from an occult pancreatic primary. J Urol Surg. [Epub Ahead of Print]

bodies. An approximately 2 cm segment of the ureter appeared fibrotic and adhered to the iliac vessels. This fibrotic segment was excised, and a left uretero-ureteral anastomosis was performed. The excised segment was sent for histopathology. This finding explained the patient's symptoms and the persistent fullness of the pelvicalyceal system, even after DJ stent placement.

Given the clinical suspicion of malignancy, tumor markers were obtained. The results revealed elevated cancer antigen 19-9 (CA19-9) (552 U/mL) and carcinoembryonic antigen (CEA) (15.5 ng/mL) levels, while alpha feto protein remained within normal limits (2.2 ng/mL). Following a multidisciplinary tumor board discussion, a positron emission tomography (PET)/CT scan was performed. This demonstrated an atrophic pancreas with multiple focal calcifications in the body and tail. A mildly fluorodeoxyglucose (FDG)-avid [maximum standardized uptake value (SUV_{max}) 3.6], suspicious soft tissue thickening measuring 16x13 mm was identified along the pancreatic tail (Figure 1). Postoperatively, the histopathology report of the excised ureteral segment revealed a well-differentiated adenocarcinoma infiltrating the ureteral wall.

Immunohistochemical (IHC) analysis of the resected specimen revealed a unique immunophenotype: The tumor cells demonstrated diffuse cytokeratin 7 (CK7) and CA19-9 expression (Figure 2), but were negative for cytokeratin 20, thyroid transcription factor 1, caudal-related homeobox transcription factor 2, special AT-rich sequence-binding protein 2, gATA-binding protein 3, paired box 8, and NK3 homeobox 1. This unusual marker profile, positive for CK7 and CA19-9 while negative for the other markers tested, raised the possibility of a pancreaticobiliary origin, which is atypical for a presumed primary ureteral adenocarcinoma.

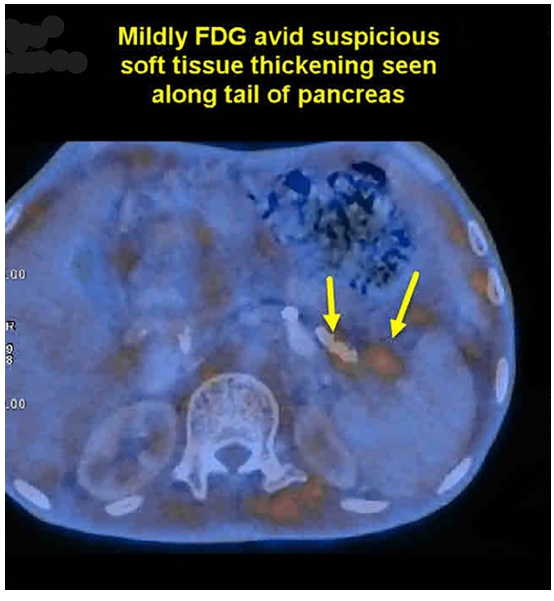

Figure 1. FDG-avid (SUV_{max} 3.6) suspicious soft tissue thickening measuring 16x13 mm was identified along the pancreatic tail

FDG: Fluorodeoxyglucose, SUV_{max}: Maximum standardized uptake value

The patient subsequently developed ascites after 2 months, leading to consideration of chemotherapy. In light of this, a laparoscopic exploration was performed. Intraoperatively, multiple metastatic deposits at the previous surgical site, multiple nodular lesions on the omentum and peritoneal surface, and moderate ascites were seen (Figure 3). Ascitic fluid was drained, and two suspicious mesenteric nodules were biopsied. Cytological examination of the ascitic fluid revealed features suggestive of adenocarcinoma. Histopathological analysis of the peritoneal nodule biopsies confirmed adenocarcinoma infiltrating fibroconnective and adipose tissue.

Discussion

Ureteral metastases are a rare occurrence, identified in only 0.3% to 8.3% of autopsies (5). Metastatic involvement of the ureter represents one of the less common causes of ureteral obstruction (3). Although malignancies originating in pelvic organs, including the prostate, cervix, and bladder, are the most frequent primary sources of ureteral tumors, pancreatic cancer metastasis to the ureter is considered exceedingly rare (6).

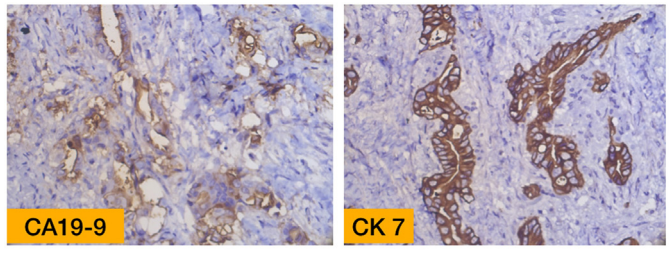

Figure 2. The tumor cells demonstrated diffuse CK7 and CA19-9 expression

CK7: Cytokeratin 7, CA19-9: Cancer antigen 19-9

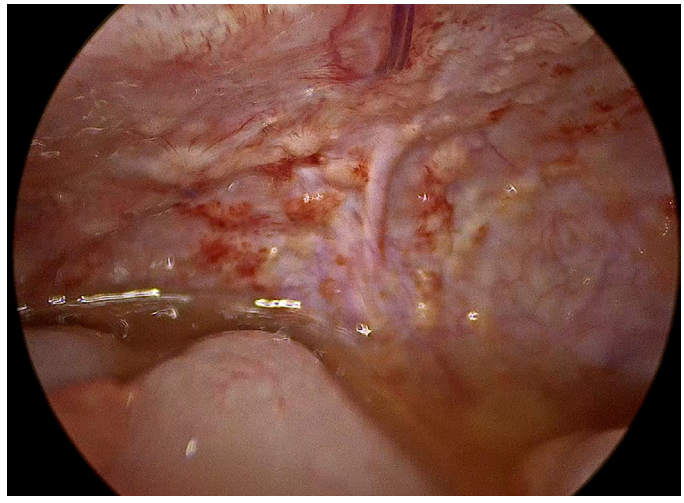

Figure 3. Multiple nodular lesions on the omentum and peritoneal surface with ascitis

Table 1. Summarizes the reported cases of ureteral metastasis from a pancreatic primary tumor, which were detected antemortem

Year	Authors	Presentation	Investigations	Course of management
1993	Roy and Baijal (2)	The patient presented with left loin pain and had left-sided ureteral involvement	Retrograde pyelography (RGP) indicated a potential ureteric stricture. Computed tomography (CT) suggestive of ill-defined infiltrative mass.	**Exploratory laparotomy revealed tough, fibrotic tissues at two left ureter sites, adherent to the psoas muscle. A large pancreatic mass was palpated. Left nephrectomy with partial ureterectomy and celiac lymph node biopsy was performed. Histopathological examination (HPE): Pancreatic adenocarcinoma.**
1993	Marincek et al. (10)	Unilateral ureteral involvement	Clinical details were not mentioned in this paper.	
1996	Stenner et al. (11) (Case 1)	Right-sided flank pain, fever. The patient underwent surgery 4 years ago for well-differentiated adenocarcinoma at the ampulla of Vater.	Ultrasonography (USG) was suggestive of right hydronephrosis. RGP: Obstruction of proximal ureter.	**Nephroureterectomy was performed. Hard nodules were palpated in the ureter. HPE: Well differentiated adenocarcinoma.**
1996	Stenner et al. (11) (Case 2)	Right-sided involvement. Case of well-differentiated adenocarcinoma of the head of the pancreas, detected 3 years back.	USG was suggestive of right hydronephrosis. Excretory urography and nephrostogram: Obstruction of proximal ureter.	**Nephroureterectomy. Hard nodules were palpated in the ureter. HPE: Well-differentiated adenocarcinoma.**
2013	Arvind et al. (4)	Right sided loin pain	The USG is suggestive of moderate hydroureteronephrosis with thinned-out renal parenchyma. CT confirmed the diagnosis. RGP: Complete lower ureteric block.	An extraperitoneal approach was taken. Three cm of the lower ureter was thickened. It was excised, and the Boari flap was constructed. HPE: Mucinous adenocarcinoma. Postoperatively, the CT scan was reviewed again, and a pancreatic lesion was detected. Biopsy revealed well-differentiated mucinous adenocarcinoma of the pancreas.
2017	Williams et al. (12)	10-day history of right abdominal pain and right flank with acute kidney injury	CT scan: Right hydronephrosis with right pelvi-ureteric junction (PUJ) narrowing. Ureteroscopy: Narrow ureteral segment distal to PUJ. The brush biopsy results were negative.	Exploration showed a fibrotic retroperitoneum with a stricture of the proximal ureter. The stenotic segment was resected. The frozen section was negative. Tension-free anastomosis was not possible; a nephroureterectomy was performed. Final HPE: Infiltrating carcinoma; immunohistochemical (IHC): Non-genitourinary source. Later, positron emission tomography (PET)/CT revealed a hypermetabolic abnormality at the head of the pancreas. Fine needle aspiration cytology confirmed adenocarcinoma of the pancreas.
2021	Verhoeven et al. (13)	Incidentally detected hydronephrosis. The patient underwent surgery 4 years ago for pancreatic carcinoma. The patient had a positive resection margin.	CT showed an obstructive soft tissue mass around the left distal ureter. The urine cytology results were negative. Biopsy of the ureteral mass showed intestinal epithelial characteristics. Molecular DNA analysis confirmed clonal relation to previous pancreatic cancer.	A nephrostomy tube was placed. The patient underwent palliative FOLFIRINOX chemotherapy.
2025	Our case	Left lower back pain and significant weight loss.	CT s/o left hydroureteronephrosis, initially managed with double J stenting. RGP: ureteric stricture. A ureteroscopy with biopsy was done. Initial biopsy: chronic inflammation.	The patient underwent retroperitoneal exploration with ureteroureterostomy. The excised suspicious segment of the ureter was a well-differentiated adenocarcinoma. PET/CT s/o: Fluorodeoxyglucose avid soft tissue thickening in pancreatic tail. IHC: adenocarcinoma arising from a pancreaticobiliary source.

Several anatomical characteristics contribute to the ureter's inherent resistance to metastatic deposits. The ureters lack a continuous network of blood and lymph vessels, making it hard for cancer cells to spread (6). Furthermore, the downward lymphatic flow of the ureter provides a countercurrent effect to the cranial lymphatic flow of the pelvic organs (7).

Despite these protective mechanisms, metastasis to the ureter can still occur. There are three major patterns of metastatic deposits in the ureter (4).

1. Periureteral adventitial infiltration (Type 1): Tumor cells often settle in the adventitia's longitudinal blood vessels and then grow into the ureteral wall. This is the most common pattern.

2. Transmural involvement (Type 2): This pattern affects the muscular, perilymphatic, or vascular layers.

3. Mucosal involvement (Type 3): This pattern is the least frequently observed and may manifest as submucosal nodules, which arise from the adhesion of tumor cells shed into the urine onto the mucosa.

Metastasis from the pancreas follows the same course as metastasis from any other organ, adhering to these established patterns. The more common Type I and Type II ureteral involvements typically present clinically as strictures, while the less frequent Type III involvement may be observed radiographically as a luminal filling defect (4,8).

Ureteral metastasis is often asymptomatic. When symptomatic, presentations typically involve obstructive features, with flank pain reported in approximately half of cases (4). Urinary tract infections are commonly observed in these patients. Hematuria is infrequent, which generally indicates the rarer occurrence of mucosal involvement (9). Ureteral obstruction secondary to metastasis typically manifests within one year of the primary cancer diagnosis. However, presentation with ureteral obstruction as the sole initial manifestation of an occult primary malignancy is exceedingly rare (2). Furthermore, most patients with ureteral metastasis exhibit widespread disease in other organs by the time of ureteral involvement (7). Antemortem diagnosis of ureteral metastasis from a pancreatic primary is exceptionally rare, with only seven cases reported in the literature. Most instances have been identified during autopsy (Table 1).

This case is unique in that ureteral involvement was the initial presenting sign of an undiagnosed primary malignancy. To identify the source of the primary, various non-invasive investigations were performed on the patient. Raised CA19-9 and CEA provided us a clue but lacked the ability to pinpoint a primary, as these tumor markers can be raised in various malignant and benign conditions (14,15).

Therefore, an FDG PET/CT was done to correlate the findings and to identify the primary malignancy. The scan suggested mild FDG-avid soft tissue thickening along the pancreatic tail with an SUV_{max} value of 3.6. According to various studies, the SUV_{max} in the case of pancreatic adenocarcinoma ranges from 3 to 10 (16). Hence, IHC was warranted to indicate the diagnosis, as an SUV of 3.6 on the FDG scan was not specific for a pancreatic malignancy. The FDG PET/CT also showed an atrophic pancreas with multiple focal calcifications. Pancreatic adenocarcinomas typically do not calcify (17). Nevertheless, they can develop in the setting of chronic pancreatitis with calcifications (18). Diffuse parenchymal atrophy, identifiable up to 60 months before the diagnosis of pancreatic adenocarcinoma, is a significant pre-diagnostic sign (19). Likewise, focal parenchymal atrophy can indicate early pancreatic cancer, although these signs are not specific for carcinoma of the pancreas (20).

IHC analysis of the tissue sample helped indicate the primary tumor in our case. The co-expression of cytokeratin 7 and CA19-9, with the negative expression of other IHC markers in the panel, indicated a malignancy of pancreaticobiliary origin (21). The IHC markers studied in our case were primarily used to indicate the origin of the primary. Immunophenotyping and genetic workup were not performed for our patient.

Ureteral stenting is frequently the initial approach and can provide long-term patency if necessary.

Nephrostomy, less commonly, ureteral reimplantation, is considered when stenting is unsuccessful (22). In this patient, given the persistent hydronephrosis despite DJ stent placement, we elected to proceed with uretero-ureteral anastomosis.

Ureteral metastasis, regardless of whether the primary tumor is known or occult, often indicates advanced disease and carries a poor prognosis (23). Available data suggest a 75% mortality rate within six months of ureteral obstruction (3).

Conclusion

Metastatic ureteral stricture is uncommon and poses challenges in identifying the primary malignancy, as demonstrated by this case. Investigations such as immunohistochemistry and PET scans are crucial tools in pinpointing the primary tumor site.

Ethics

Informed Consent: Informed consent was obtained from the patient.

Footnotes

Authorship Contributions

Surgical and Medical Practices: S.J., K.R., V.P., R.C., Concept: K.R., R.C., Data Collection or Processing: S.J., Analysis or

Interpretation: K.R., Literature Search: V.P., Writing: S.J., K.R., R.C.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

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